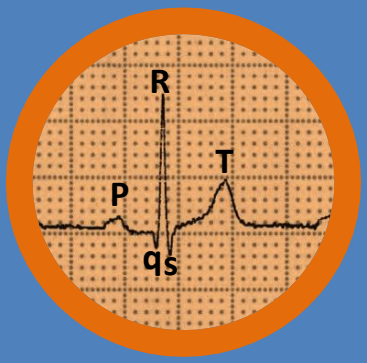

ECG Review : With Pulmonary disorders

(For Academic Purpose only)

Dr. DURGA PRASAD KHAITAN

MD (MEDICINE) FCGP (IND) FIAMS (MEDICINE) FICP FICCMD FIACM

Each wave on ECG tells a tale so precise
In pulmonary disorders it adds to our surprise .....

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Each wave on ECG tells a tale so precise
In pulmonary disorders it adds to our surprise …..

Knowledge and skill in the field of electrocardiography are constantly changing with the new researches and understanding.

With humble words I wish to say that some momentous articles with ECG changes in pulmonary disorders have been covered within this ebook. It is only a step towards the vast ocean of knowledge. I may be excused for any error or omission.

With thanks and regards

DEDICATED
TO
FELLOW COLLEAGUES

Index

ELECTROCARDIOGRAPHIC CHANGES IN CHRONIC OBSTRUCTION PULMONARY DISEASE

ECG

ELECTROCARDIOGRAPHIC CHANGES IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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OUTLINE

Introduction

Mechanism of ECG changes

- ☐ Insulating effect with low voltage QRS
- ☐ Hypoxic drive with pulmonary vasoconstriction and pulmonary hypertension. Destruction of pulmonary tissue with loss of pulmonary capillaries also contributes to pulmonary hypertension.
- ☐ Clockwise rotation in the transverse plane , with pushing of the right ventricle anteriorly and the left ventricle somewhat posteriorly

Associated electrocardiographic events in COPD

- ☐ Low voltage QRS complex
- ☐ Pulmonary hypertension leads to
  - Overload over the right ventricle reflected as right ventricular hypertrophy (RVH)
    ↓
    Overload over the right atrium reflected as right atrial enlargement (RAE)
- ☐ The clockwise rotation
  R/S ratio<1 in leads V5/V6 ; Poor R wave progression
- ☐ Associated arrhythmias
- ☐ Miscellaneous ECG findings
  - Lead I sign (Schamroth's sign)
  - SI, SII, SIII pattern

Very useful Chou's criteria suggesting the presence of COPD

Illustration by ECGs

Take Home Message

References

Electrocardiographic changes in chronic obstructive pulmonary disease

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A Narrative Review

Lungs and heart are interconnected with each other. They avail happy moments of life by remaining together throughout. Two grow together , live together and share together. One suffers , the other suffers – so is the twin friendship.

In COPD the heart shares the agony – unbreakable twin bond , one reflects the other.

- ☐ **The resultant hypoxic drive in COPD with accompanying destruction of the lung tissue with loss of pulmonary capillaries results in pulmonary hypertension , with superadded overload onto the right side of the heart.**
- ☐ **Having its fixed attachment to the great vessels the hyperinflated lungs together with ↑RV mass cause clockwise rotation in the transverse plane, with the movement of the right ventricle anteriorly and displacement of the left ventricle posteriorly.**

The clinicians peep through the ECG and access COPD sufferers in the light of clinical findings and ECG both.

1. Introduction

Chronic obstructive pulmonary disease is a progressive disorder characterized by airflow limitation / obstruction – either not reversible at all or only partially reversible. Chronic bronchitis and emphysema are classified together as COPD. The presence of airflow limitation by premature airway closing in chronic bronchitis and the enlarged air spaces in emphysema with pulmonary tissue destruction are the main culprit. There is pulmonary hypertension either by hypoxic drive or by the loss of pulmonary capillaries as a part of destructive process.

The prevalence of COPD is very common all over the globe , usually caused by increasing tobacco consumption and air pollution. This would be very helpful to study electrocardiographic changes in COPD , which add to its diagnostic assessment with prognostic implications as well. In patients with COPD , the resultant pulmonary hypertension increases RV afterload with more RV mass , which predisposes to clockwise rotation in the horizontal plane , with the reduction in the QRS amplitudes as well.

2. Mechanism of ECG changes

- ☐ **Insulating effect** : low voltage QRS due to the overlying hyperinflated lungs in between the heart and the exploring electrodes.
- ☐ **Hypoxic drive** : This causes pulmonary vasoconstriction with pulmonary hypertension resulting in right atrial and right ventricle hypertrophy (Cor-pulmonale). Destruction of pulmonary tissue with loss of pulmonary capillaries further increases the resistance of the pulmonary vascular bed by reducing the effective surface area .

☐ Having its fixed attachment to the great vessels the resultant increased right ventricular mass in association with hyperinflated lungs causes **clockwise rotation in the transverse plane** , with pushing of the right ventricle anteriorly and the left ventricle somewhat posteriorly.

In COPD

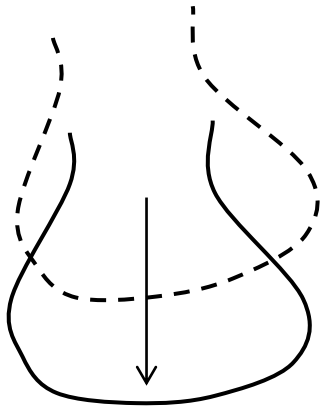


Fig. 1.1

- Hyperinflated lungs lie in between the heart and the exploring electrodes.
- These hyperinflated lungs with flattening of the diaphragm also displace the heart downward – the heart adopts a longitudional tubular position. Right ventricular hypertrophy attributes much to such a shift rather than the hyperinflated lungs alone – as viewed by **Obasohan et al.**

In 2011 , Obasohan et al demonstrated very clearly in their study group that a shift of electrical axis of the heart occurring in COPD is mainly due to the development of right ventricular hypertrophy (Cor-pulmonale) , rather than the hyperinflation of lungs itself.

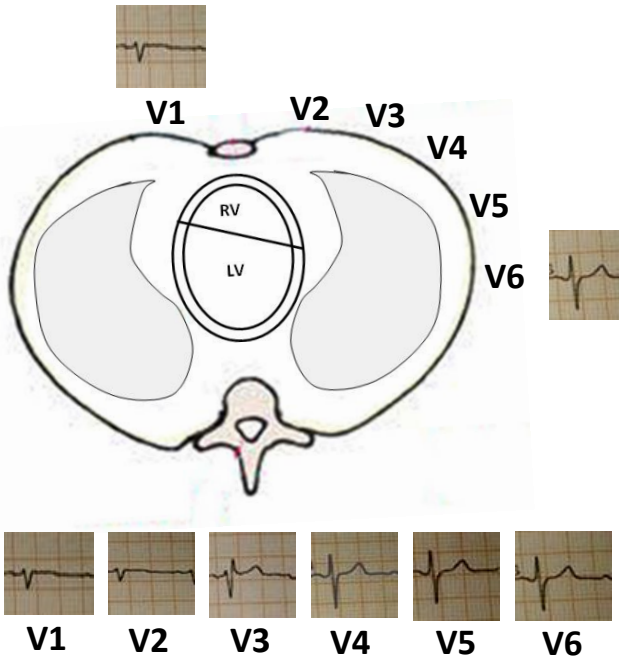


Fig. 1.2

Clockwise rotation

- Having its fixed attachment to the great vessels the heart also undertakes clockwise rotation in the transverse plane , with the movement of the right ventricle anteriorly with the displacement of the left ventricle posteriorly, thereby both the ventricles lie more or less parallel to each other. And so the right ventricle assumes the more anterior position.
- This Clockwise rotation is manifested as R/S ratio <1 over the chest leads extending even upto V5/V6
- Placing of the precordial leads onto supposed basal cardiac zone, which has been dragged down also superadds to the clockwise rotation

3. Associated electrocardiographic events in COPD

ECG findings in COPD may be summarized , as illustrated within the following table :

- ☐ The voluminous lungs encroaching upon the heart
- ☐ Pulmonary hypertension with its consequences
- ☐ The Clockwise rotation
- ☐ Associated arrhythmias
- ☐ Miscellaneous ECG findings

☐ **The voluminous lungs encroaching upon the heart** is having an insulating effect – diminishing the transmission of electrical potential recorded through the exploring electrodes resulting in low voltage QRS.
QRS amplitude may be registered in all the limb leads <0.5 mV (5mm) / <1.0 mV (10 mm) in the precordial leads.

☐ **Pulmonary hypertension** → overload over the right ventricle and the right atrium (right ventricular pressure is being transmitted to the right atrium).

- **Overload over the right ventricle reflected as right ventricular hypertrophy (RVH)**
  - ➢ The most reliable sign of RVH in the presence of COPD is right axis deviation and dominant S waves over left precordial leads ; others are right atrial enlargement (indirect evidence) , and RSR' or qR pattern in lead V1 with ± inverted T waves in right precordial / inferior leads.
    (RSR' pattern indicates the extension of right ventricular hypertrophy upto the right ventricular outflow tract ; qR pattern is usually associated with a right ventricular pressure that is greater than the left ventricular pressure**)**.
  - ➢ RBBB due to the delayed activation of more dilated /hypertrophied right ventricle.
  - ➢ The ECG looses its value in the diagnosis of RVH in the presence of coexisting LVH or myocardial ischemia.
- **Overload over the right atrium reflected as right atrial enlargement (RAE).**
  - ➢ P >0.25 mV (P-pulmonale the most common ECG abnormality) in leads II , III , aVF / P as a +ve initial wave in V1>0.15 mV.
  - ➢ P axis deviation towards the right side over the frontal plane ($+60^0$ to $+90^0$). (Normal P axis = 0^0 to $+75^0$)

Baljepally & Spodick confirmed in study of 100 patients with vertical P axis (>70 degree) having high sensitivity (89%) and high specificity (96%) as a sign of pulmonary emphysema.

NB : The frontal T wave axis may follow the rightward deviated QRS axis resulting in tall T wave in lead III than in lead I. Otherwise , T waves are of low amplitude. The T wave inversion may be seen in right precordial leads due to associated right ventricular hypertrophy.

(Generalized ST segment depression / T wave inversions might occur due to associated global hypoxemia).

☐ **The Clockwise rotation is responsible for the following electrical events on ECG :**

- R/S ratio <1 to be observed even upto leads V5/V6.
  (Here to mention again that the most reliable sign of right ventricular hypertrophy in COPD is deeper S waves over left precordial leads in association with right axis deviation).
- There may be SV1 , SV2 , SV3 pattern (complete absence of R wave in V1-V3 due to the orientation of precordial leads upwards towards the basal region due to the downward displaced heart).
- Poor R wave progression over the chest leads from V1-V6, also due to the orientation of the precordial leads upwards.

☐ **Associated arrhythmias**

Supraventricular arrhythmias are more commonly encountered compared to ventricular arrhythmias. The ventricular arrhythmias usually occur in the form of multifocal VPCs. Multifocal atrial tachycardia is almost pathognomonic of the presence of pulmonary insufficiency. Other arrhythmias are – atrial fibrillation (most common arrhythmia in COPD) , atrial tachycardia , atrial flutter (less often).

Most of the patients are having evidence of RVH with increased evidence of ventricular ectopi observed during sleep in the presence of oxygen desaturation.

Sinus tachycardia may be observed during acute exaggeration. Resting heart rate is increased with severity of COPD , associated with both cardiovascular and all-cause mortality.

☐ **Miscellaneous ECG findings**

- **Lead I sign (Schamroth's sign)**

  In 1965, Fowler et al proposed very strict criteria for the diagnosis of lead I sign on ECG , consisting of isoelectric P wave in lead I combined with a very small QRS complex of less than 1.5 mm as a total deflection and T wave of less than 0.5 mm.

  This sign was observed in this group of researchers to be present in 33% of severe pulmonary emphysema with Cor-pulmonale patients.

  Further Schamroth modified the lead I sign being reflected on ECG as 'absent or very low amplitude P , QRS ,T wave complexes in lead I giving the appearance of a minimally disturbed baseline' without any specification of the cut off values for the amplitude of these three wave forms.

  The 'lead I sign' is a highly specific ECG marker of chronic obstructive pulmonary disease – rarely being demonstrated in any other condition. The diagnosis of chronic obstructive pulmonary disease should be strongly suspected when one sees this lead I sign on ECG. This is to be mentioned here that with poor resource-setting in emergency , this sign would be very helpful in the diagnosis of the patients with COPD.

Broadly to say , lead I sign is due to more or less vertically placed heart having all P, QRS, T axes being oriented almost perpendicular (round about 90^0) to lead I , friendly with the rule of electrical axis , **as per rule of 90^0.**

Rule of electrical axis : Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive current , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection.

Thus , electrical flow is having either small / nil deflection or an equiphasic response with ECG waves in a lead placed at 90^0 away with reference to the vector.

- **The SI, SII, SIII pattern** in standard leads I, II and III causing shifting of the QRS axis to the region of northwest zone (-90^0 to -150^0) reflects the conduction delay in the right ventricle with posterior displacement of the apex. This pattern is an expression of late or terminal forces of ventricular depolarization that are directed superiorly and to the right. This pattern also indirectly indicates the presence of associated RVH pushing the cardiac apex somewhat posteriorly.

- **Prominent atrial repolarization**

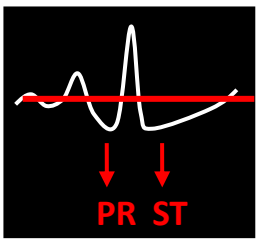


Fig. 1.3

RAE→ A stronger electrical signal during repolarization → part of Ta wave on its either side comes out, usually Ta wave is hidden behind the corresponding QRS complex → **seen as sagging of PR segment also with ST segment , being dragged downward** (i.e.stronger atrial repolarization wave lowers the baseline) , best seen in inferior leads – mainly lead II.

4. Very useful Chou's criteria suggesting the presence of COPD

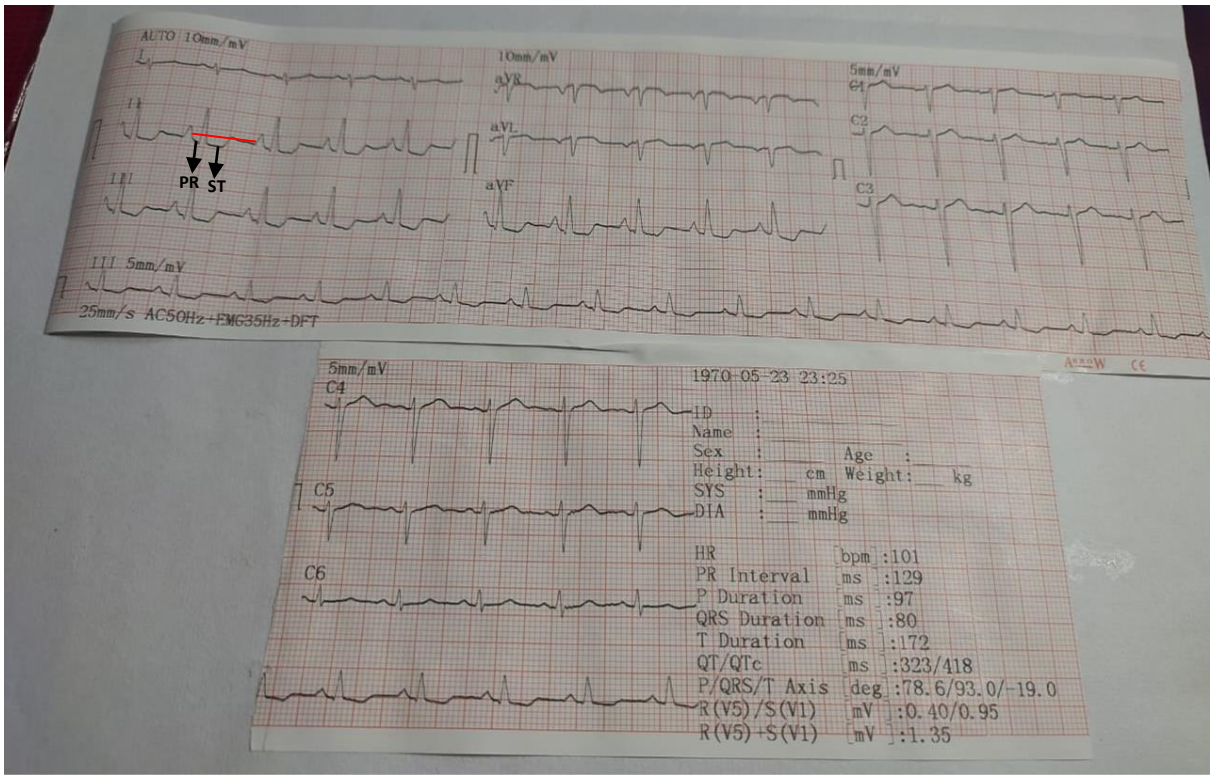
According to Chou , COPD is likely to be present if one or more of the P wave changes with one or more of the QRS changes as enumerated below , are present on ECG:

P wave changes	1. P waves > 0.25 mV in lead II, III, aVF. 2. P wave axis to the right of 80 degrees in the frontal plane. 3. Lead I sign with an isoelectric P wave , QRS amplitude <0.15 mV, and T wave amplitude <0.05 mV.
QRS changes	4. QRS amplitude in all limb leads <0.5 mV 5. QRS axis to the right of 90 degrees in the frontal plane. 6. QRS amplitude <0.5 mV in lead V5 or V6; or R wave <0.7 mV in lead V5 or R wave <0.5 mV in lead V6. 7. R/S ratio < 1 in lead V5 or V6. 8. $S_1S_2S_3$ pattern with R/S ratio <1 in leads I, II and III.

5. Illustration by ECGs

ECG 1 :

44 years male – a case of COPD



Source : CME INDIA (28.05.2022) by Dr. Pradeep Sahay , Senior consultant Physician , Giridih

ECG findings

- Heart rate 100 bpm
- Limb leads
  - QRS axis $+110^0$ (right axis deviation)
  - P axis $+75^0$ with P-pulmonale in lead II , III and aVF.
  - Sagging of PR segment with ST segment dragging downward (compared with isoelectric Tp segment)
  - Minimum deflection P-QRS-T in lead I , compared to other limb leads (possibly lead I sign)
- Chest leads
  - Poor R wave progression (<3mm in V3)
  - Clockwise rotation with R/S ratio < 1 in lead V5

Discussion

☐ The combination of right axis deviation with dominant S wave over precordial leads (V1-V5) with R/S ratio <1 in lead V5 is very much suggestive of right ventricular hypertrophy (RVH)

☐ P axis = + 75^0

Baljepally & Spodick confirmed in study of 100 patients with vertical P axis (>70 degree) having high sensitivity (89%) and high specificity (96%) as a sign of pulmonary emphysema.

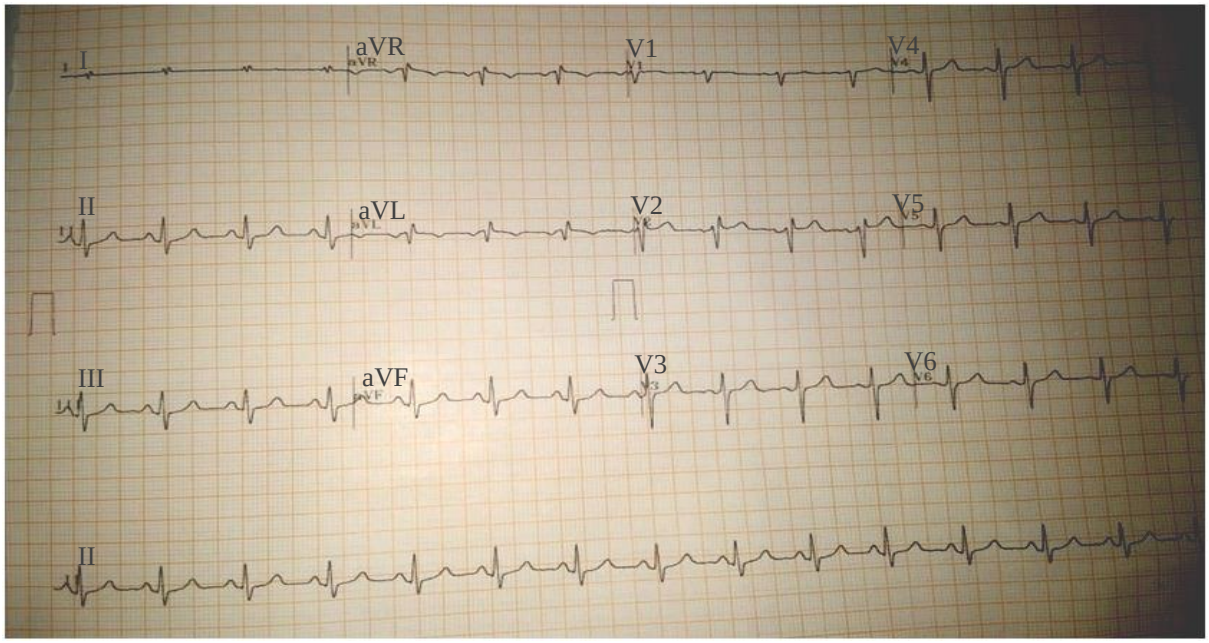
Continued over the next page …

- ☐ Sagging of PR segment with somewhat ST segment dragging downward reflects atrial repolarization to be prominent.
- ☐ Minimum deflection of P-QRS-T in lead I , compared to other limb leads is most possibly due to lead I sign (Schamroth's sign)
  (Schamroth modified the lead I sign being reflected on ECG as 'absent or very low amplitude P , QRS ,T wave complexes in lead I giving the appearance of a minimally disturbed baseline' without any specification of the cut off values for the amplitude of these three wave forms)

The findings mentioned above are also consistent with Chou's criteria of COPD (please see Page No 6)

ECG 2

70 years old male admitted for cough , fever , breathlessness of 5 days duration ; history of recurrent LRTI; no h/o DM , CAD ; stopped smoking recently.



Source : CME INDIA (16.03.2020) by Dr. SS Lakashmanan , Senior consultant Physician , Chennai

ECG findings :

- Over the limb leads
  - Lead I sign : isoelectric P being merged with the baseline combined with QRS complex of <1.5 mm and T wave <0.5 mm (also consistent with Schamroth modified lead I sign)
  - Rightward P axis at $+90^0$
- Over the precordial leads
  - Clockwise rotation : R/S <1 in chest leads including V5 and V6 both.
  - R wave <0.5 mV in lead V6.

Discussion : As per Chou's criteria , two P wave changes (lead I sign + rightward shift of the P wave) and two QRS changes (R/S ratio <1 in lead V5 and V6 + R wave <0.5 mV in lead V6) are in favour of COPD.

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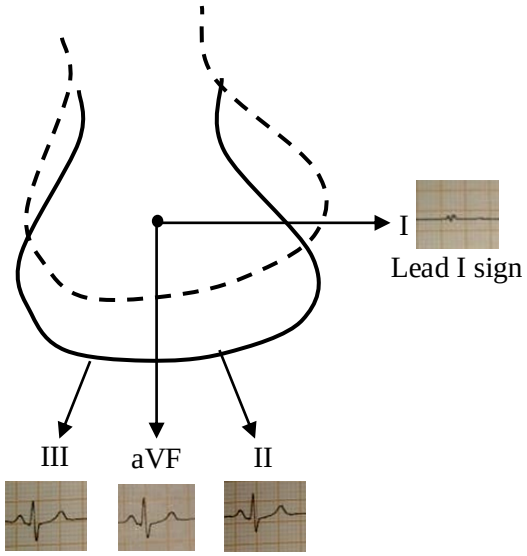

P wave being the smallest in size amongst all the waves , is recorded on ECG as nil deflection represented by isoelectric line. Since the electrical forces axes of P , QRS, T run longitudinally very close to 90^0 , the inferior leads II, III and aVF are rather having the obvious visible display of all these waves on ECG but the reverse events with lead I.

Fig. 1.4

6. Take Home Message

- ☐ **Low voltage QRS complex**
  In all limb leads <0.5 mV (5mm) / <1 mV (10 mm) in all the precordial leads
- ☐ **Clockwise rotation** with pushing of the right ventricle anteriorly and the left ventricle somewhat posteriorly (R/S ratio <1 over the chest leads even upto V5/V6).
- ☐ **Pulmonary hypertension** on ECG is reflected as through RVH.
  - RVH
    - ✓ Right axis deviation + R/S ratio <1 in V5/V6 (the most reliable sign)
    - ✓ RSR' or qR pattern in lead V1 with ± inverted T waves in right precordial/ inferior leads.
    - ✓ The SI, SII, SIII pattern in standard leads I , II and III
  - Right atrial enlargement (RAE) : Indirect evidence
    P>0.25 mV (p-pulmonale the most common ECG abnormity) in leads II, III ,aVF / P as a +ve initial wave in V1>0.15 mV.
    P axis deviation towards the right side over the frontal plane
    (Normal P axis $=0^0$ to 75^0)
- ☐ **Associated arrhythmias**
  - Multifocal artial tachycardia is almost pathognomonic of the presence of pulmonary insufficiency. Other arrhythmias are – atrial fibrillation (most common arrhythmia in COPD) , atrial tachycardia , atrial flutter (less often).
  - Ventricular ectopi observed during sleep in the presence oxygen desaturation.
- ☐ **Miscellaneous ECG findings**
  - Lead I sign (Schamroth's sign) : Minimum deflection of P-QRS-T in lead I, compared to the other limb leads // Also to note SV1,SV2, SV3 pattern on ECG.
  - Prominent atrial repolarization : sagging of PR segment with somewhat ST segment dragging downward reflects atrial repolarization to be prominent.

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ECG CHANGES IN ACUTE PULMONARY EMBOLISM

ECG

ECG CHANGES IN ACUTE PULMONARY EMBOLISM

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OUTLINE

Introduction

Acute pulmonary embolism presents itself with variable changes on ECG - Sinus tachycardia being the commonest

Pathophysiological Basis

Overstimulation of sympathetic nervous system → sinus tachycardia
Increased RV pressure → RV ventricular strain , RV and RA dilatation, clockwise rotation
Increased myocardial oxygen demand →myocardial ischemia

Associated ECG changes

- Sinus tachycardia
- RV strain due to sudden increase in pulmonary vascular resistance
- RV dilatation and its impact on ECG
- Myocardial ischemia
- Miscellaneous findings

Illustration by ECGs

Take Home Message

References

ECG changes in Acute Pulmonary Embolism

A Narrative Review

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The Fire of Pulmonary embolism is the long threaded , set blazing high , it also touches the gated house of the Heart. Being a violent overthrower , it sends the signals also to the heart.

- ☐ **The most common is sinus tachycardia attributed to overstimulation of the sympathetic nervous system due to associated pain , anxiety and hypoxia**
- ☐ **Right ventricular strain as reflected on ECG by T wave inversion over the right precordial leads (V1-3, even upto V4) ± inferior leads (II, III , aVF) is the very significant finding in acute pulmonary embolism**

The clinicians catch the high-kindled signals and extinguish thy fire by their best efforts.

1. Introduction

Acute Pulmonary embolism presents itself with variable clinical manifestations and it should always be kept in mind as a differential approach for acute shortness of breath , chest pain , or even syncope. The ECG remains most widely used and easily available tool.

Though the ECG often picks up the abnormalities associated with pulmonary embolism but the findings are neither sensitive nor specific to reach to the diagnosis or exclude PE. **This is to be noted that 20% of patients with Pulmonary embolism are having a completely normal ECG**. The ECG changes consistent with acute pulmonary embolism may be observed in any condition causing acute pulmonary hypertension including hypoxia causing pulmonary hypoxic vasoconstriction. The additional utility of the ECG recording in such a demanding situation is also ruling out the other devastating life-threatening conditions like acute myocardial infarction.

2. Pathophysiological Basis

Acute Pulmonary embolism is reflected on ECG as attributed to the following factors :

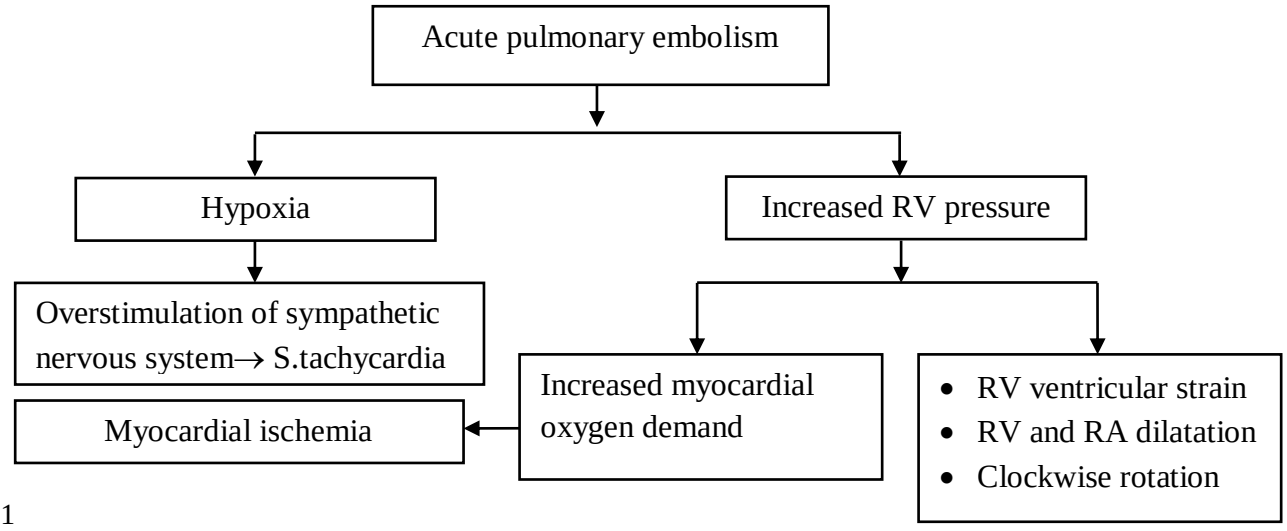


Fig. 1.1

Such changes over the right ventricle , as enumerated may lead to reduced RV stroke volume with resultant reduced left ventricular preload and reduced coronary perfusion as well. Thus ischemic changes might also be extended even to the left ventricle.

3. Associated ECG changes

A good number of patients – upto 20% or more may not reveal any remarkable electrocardiographic abnormalities , as a result of the embolic phenomenon. Many of the ECG abnormalities do appear very early , say within minutes to few hours at the onset of the event. Therefore , it becomes very essential to pickup these abnormalities of earlier onset by recording the ECG changes as early as possible (within 1-2 hours) and afterwards , serial ECG changes should be recorded to see the progressive changes.

ECG changes are rather dynamic in nature.

These changes can be explained and listed as follows :

- **Overstimulation of the sympathetic nervous system** due to pain, anxiety and hypoxia

  ↓

  Sinus tachycardia

  The most common ECG denominator in the event of acute pulmonary embolism is sinus tachycardia in about half of the cases.

- **RV strain due to sudden increase in pulmonary vascular resistance (34%)**

  - T wave inversions over the right precordial leads (V1-V3 , even upto V4; more significant if noticed over > 2 such precordial leads) ± the inferior leads (II, III , aVF).

    An ECG showing RV strain in an acutely breathless patient is highly suggestive of acute pulmonary embolism.

  - **'$S_1Q_3T_3$' pattern** (McGinn-White Sign)

    In 1935 McGinn and White firstly presented his clinical paper correlating ECG findings in five patients within 21 hours of the clinical events of pulmonary embolism showing a prominent S wave in lead I , Q wave in lead III and negative T wave in lead III.

    This sign implies acute pressure and volume overload over the right ventricle as a consequence of pulmonary hypertension – reflecting right ventricular strain. Any cause of acute cor pulmonale , such as pulmonary embolism , acute bronchospasm , pneumothorax , any other acute lung disorder , etc. might be responsible for this sign. This is neither sensitive nor specific for pulmonary embolism and this sign is not always present on ECG.

    A large S wave in lead I , Q wave in lead III and an inverted T wave in lead III - a pointer better to say towards right ventricular strain.

    The prominent S wave (a deep and narrow deflection) in standard lead I is due to acute RV strain. This S wave usually occurs earlier within 24 hours and returns to the normal gradually over a number of weeks.

The Prominent Q wave in lead III - Q wave is less than 0.04 sec in duration and the magnitude is less than 25% of the following R wave ; a QS complex is not encountered with acute pulmonary embolism.

The anatomical concept : To mention here that the right ventricle occupies about one-third of the inferior (diaphragmatic) surface of the heart while the left ventricle occupies roughly two-thirds of the inferior surface.

This Q wave might occasionally also be seen in aVF or even standard lead II with severe right ventricular dilatation , pushing it more towards the left.

T wave inversion in standard lead III - This indicates shifting of the T wave axis towards the left , 90^0 leftward away from the lead III. Sometimes T wave inversion in lead aVF with an equiphasic T wave in standard lead II might be in association.

RV dilatation and its impact on ECG

- **Complete or incomplete right bundle branch block (RBBB)** in 18% of such cases due to the stretched-pressure of the dilated and distorted right ventricle.
- **Right axis deviation** in 16% of such cases. This may be associated with S wave in lead I (a rare but possible 'Pseudo left axis deviation' might be seen secondary to a marked right ventricular strain).
- **Dominant R/QR wave in V1** (Right ventricular dilatation)
- In some cases, there may be **right atrial enlargement** (as P pulmonale on ECG) due to the transmitted pressure from below dilated right ventricle. Right atrial dilatation may also be attributed to increased tricuspid regurgitation resulting from increasing RV dilatation.
- **Clockwise rotation** - with shift of the transitional zone towards the left , even extending upto lead V5 or V6. This occurs due to the RV dilatation.

Myocardial ischemia

- Associated with elevation and coving of the ST segment over the right precordial leads , attributed to epicardial injury simulating with acute anteroseptal infarction
- The ST segment depression in leads I and II may occur. A 'stair-case' ascent of ST segment (flattening of the initial part of ST segment and T-wave , followed by a more or less sharp rise and then flattening of terminal portion of the T wave) may be seen.

Miscellaneous findings

- Low amplitude deflections – In some cases this occurs as a tendency to low voltage on ECG, the cause being uncertain.
- Atrial tachyarrhythmias in some cases – Atrial extrasystoles , atrial fibrillation , atrial flutter , atrial tachycardia.

NB : The findings of right heart strain and atrial arrhythmias are having a bad prognosis.

4. Different diagnostic scoring system by different workers

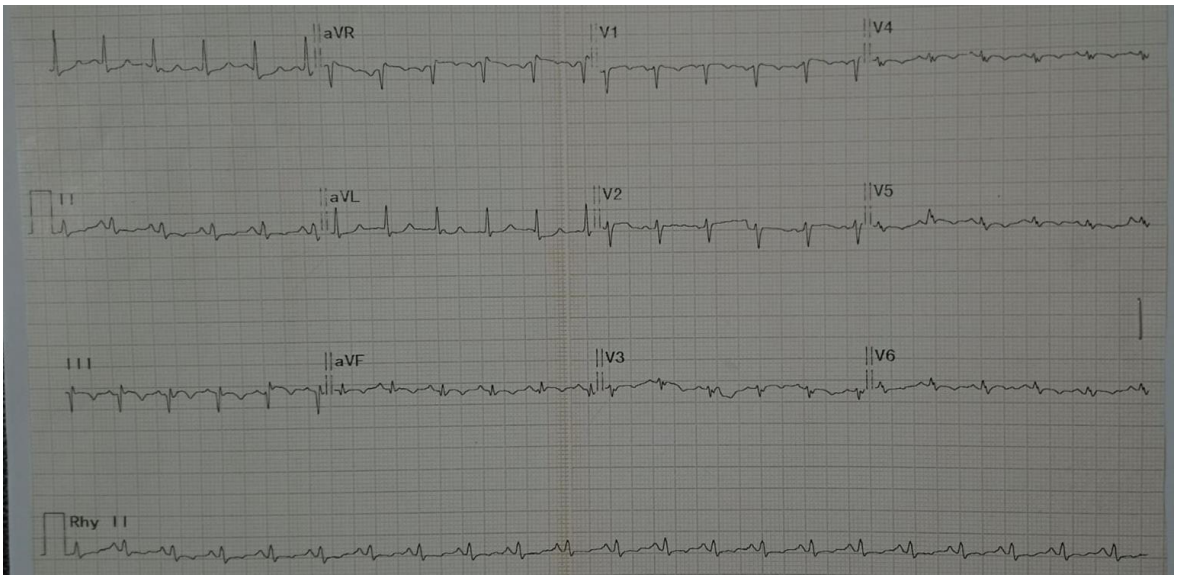
- **Sukhija's et al**. showed the presence of the two of the following five ECG abnormalities S1 , Q3 , S1Q3T3, Sinus tachycardia , supraventricular arrhythmia as associated with a specificity of 96 % and a positive predictive value of 94%.
- **Sreeram et al**. 76% probability if 3 or more of the following features are present.

I. Incomplete or complete RBBB which was associated with ST segment elevation and positive T wave
II. S wave in lead I and aVL>1.5 mm
III. A shift in the transition zone in the precordial leads to V5
IV. Q waves in Leads III and aVF, but not in lead II
V. RAD of the QRS in the frontal plane
VI. Low-voltage QRS complexes <5 mm in the limb leads
VII. T wave inversion in Leads III and aVF or Leads V1 to V4

The sensitivity of Sreeram's predictive rule has not been validated by other studies ; however , they have shown it to be quite specific (94.2%).

5. Illustration by ECGs

ECG No. 1 : 48 years old obese diabetic female with chest pain and breathlessness (DVT both lower limbs)



Source : Dr. S S Lakshmanan , Senior consultant physician , Chennai

Findings :

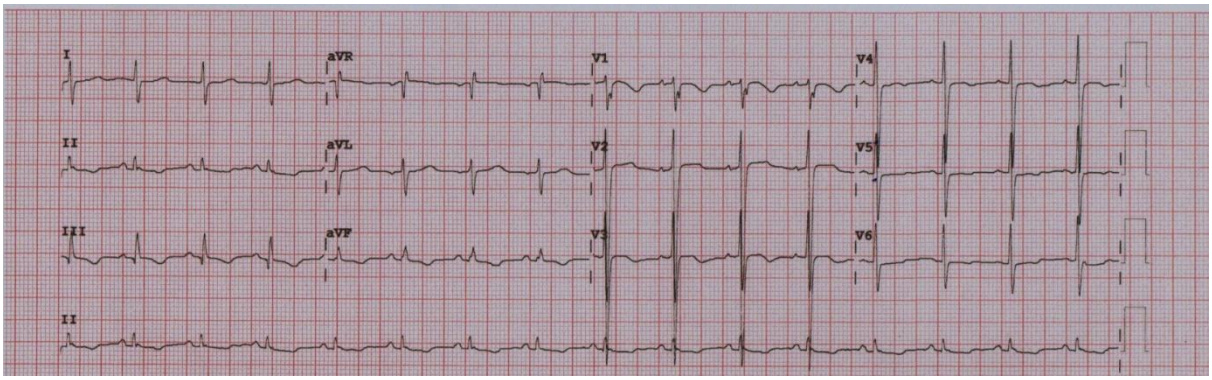
- Sinus tachycardia
- T inversion in leads II , II , aVF and V1 , V3-V6 (indicating right ventricular strain with reduced coronary perfusion over LV) , S1Q3T3 is questionable due to the absence of large S-wave in lead I
- Clockwise rotation
- Low voltage QRS over chest leads

Relevant investigations :

- D Dimer 6230 with CT PUL ANGIO Positive

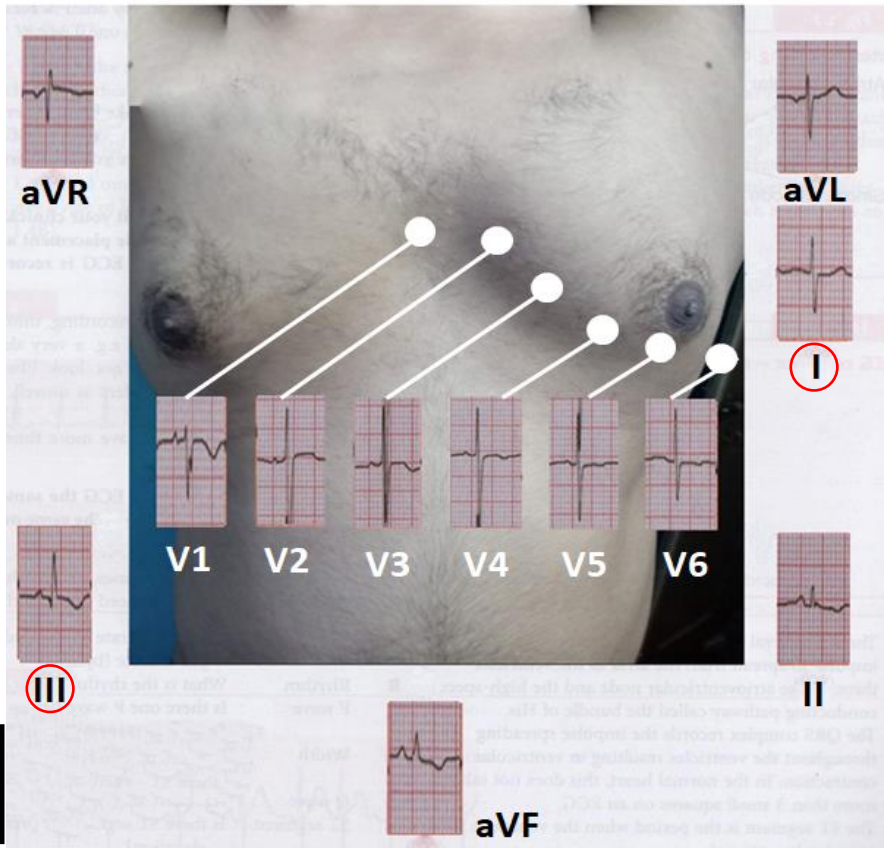
ECG No. 2

A 42 years male suddenly developed mild palpitation with the following ECG :



Findings :

- Mild Sinus tachycardia
- T wave inversion over V1 and V3 , a tendency to have T wave inversion over V2. T wave inversion also over inferior leads (II, III, aVF).
- $S_1Q_3T_3$ Pattern
- Right axis deivation
- Clockwise rotation



S1 Q3 T3 Pattern

Fig. 1.2

CARDIAC ECHO OF THE SAME PATIENT with the following findings :

Interpretation Comments
RA/RV/MPA DILATED. NO RWMA OF LV. 2DLVEF = 55% MILD TR, PASP=58 MMHG.TAPSE = 16 MM. NO MS/ASD/PS. NO MR/AR/COA. IVC = 17 MM &<50% COLLAPSIBLE.
FINAL IMP : MILD TR, MOD PAH, NORMAL BIVENTRICULAR FN.
D/D 1. PPH. 2. PULM EMBOLISM
TO CORRELATE CLINICALLY

CT PULMONARY ANGIOGRAPHY OF THE SAME PATIENT (Pulmonary Angiogram is a Gold-Standard providing more or less 100% certainty that an obstruction exists in the pulmonary artery ; negative angiogram provides > 90% certainty in the exclusion of PE)

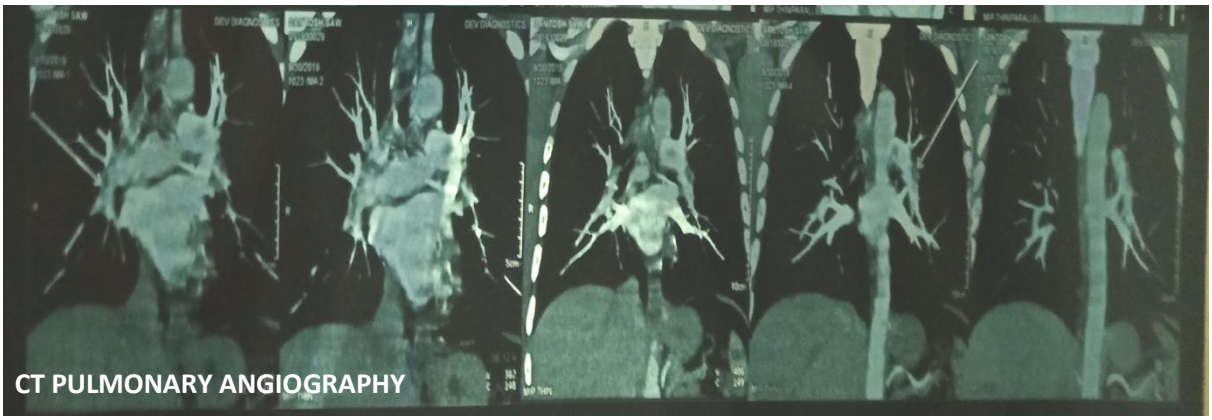


Fig. 1.3 **Thrombus in right pulmonary and left lower lobe pulmonary arteries causing partial filling defects – Pulmonary embolism.**

6. Take Home Message

- ☐ 20% percent cases with pulmonary embolism have a complete normal ECG .
- ☐ To record ECG changes as early as possible (within 1-2 hours) after the onset of relevant symptoms , then serial ECG changes to be recorded to see the progressive changes.
- ☐ The commonest change on ECG is sinus tachcycardia (in about half of the cases)
- ☐ RV strain due to sudden increase in pulmonary vascular resistance
  - T-wave inversion over V1-V4 $\pm$ over the inferior leads
  - S1Q3T3 pattern (McGinn-White Sign)
- ☐ RV dilatation and its impact on ECG :
  RBBB , Right axis deviation , dominant R/QR wave in V1, RAE , clockwise rotation
- ☐ Myocardial ischemia
  - Coved ST↑ over right chest leads (epicardial injury)
  - ST segment depression in lead I and II – 'Stair-case' ascent , as discussed in page no. 15
- ☐ Miscellaneous findings : Low voltage QRS , atrial tachyarrhytmias in some cases

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THE SI SII SIII SYNDROME ON ECG

ECG

THE SI SII SIII SYNDROME ON ECG

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OUTLINE

The SI SII SIII Syndrome on ECG

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A Narrative Review

A paradigm shift at times in life becomes essential – a move more towards a comfortable and safety adaptation. This proves to be a safety essential for survival. I call this an axial shift of life , which allows a human race to survive.

The SI SII SIII syndrome is one of the examples of such a paradigm shift on ECG.

- ☐ The term 'SI SII SIII' syndrome refers to an ECG pattern where there are prominent S waves in leads I , II , III. This is not a specific marker for a single entity but rather an indicator of right heart overload or even strain.
- ☐ The enlarged or strained right ventricle produces more electrical activity which shifts the electrical axis more superiorly and rightward – this shift may be called as a new adaptation for the survival even when the heart remains firmly anchored to the underneath diaphragm by the central tendon.

The presence of SI SII SIII pattern on ECG prompts a clinician in the search of its root cause leading to right ventricular overload or strain.

1. Introduction (keynotes)

- As its name implies SI SII SIII syndrome is an electrocardiographical expression with prominent S waves in leads I II III with a positive deflection in lead aVR -------- the result of superior and rightward shift in the heart's electrical axis
- To make it simple , when the right ventricle is hypertrophied or strained , it becomes electrically dominant over the left ventricle , causing the heart's electrical axis to be shifted more rightward and upward→ this depolarization shift is away from leads I II III → prominent S waves in these leads.
  (Negative waves inscribed in those leads which are away from the electrical axis**)**

Superior and rightward shift of Electrical axis (-90^0 to -150^0)

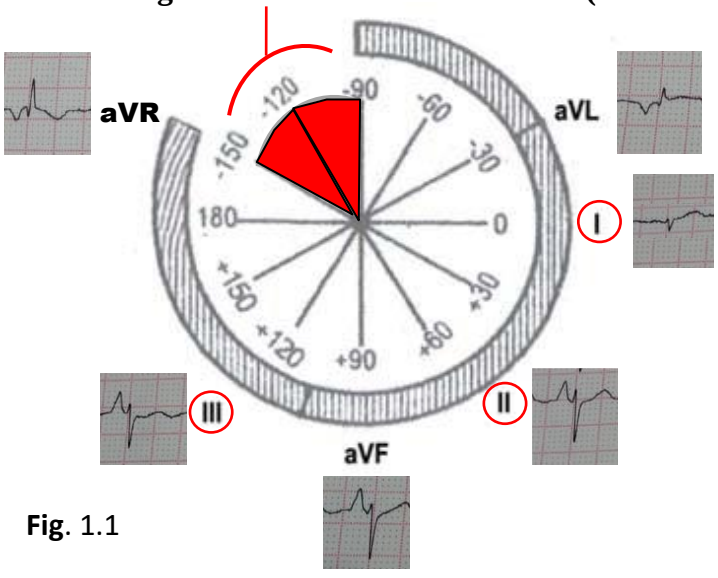


Fig. 1.1

Prominent S-waves in leads I II III with a positive deflection in lead aVR

- This pattern was initially brought to the light in 1960 by Burch and D Pasqulae in association with ventricular septal defect and shortly thereafter in adults with chronic obstructive pulmonary disease.
- The SI SII SIII syndrome is not a specific marker for a single entity as such but rather indicative of right ventricular overload or strain. It is not an uncommon electrocardiographic finding , seen usually associated with chronic obstructive pulmonary disease (eg. COPD)
- The presence of such findings on ECG prompts further investigations directed towards the underlying cause , those including echocardiography , chest radiology or at times even CT imaging to access the right heart and pulmonary vasculature.

2. Electrophysiology (Mechanism)

In a normal heart , the electrical axis is typically pointing downwards and to the left , reflecting the dominance of the left ventricle. This results in positive QRS complexes (upright R-waves) in leads I and II , which are oriented towards the left.

✓ But the enlarged or strained right ventricle sets its electrical signal more rightward and upward as a result of more electricity production attributed to the altered anatomical remodelling of the heart.

The basic reasoning behind SI SII SIII syndrome is summarized as below :

> The basic reasoning of this SI SII SIII seems to be related to somewhat delayed right ventricular depolarization due to remarkable right ventricular hypertrophy or secondary to its acute strain, shifting the electrical axis more right and upward.
> Only simple RVH or its strain might have produced only rightward shift , not so directed upward

The electrocardiographic findings have been well illustrated in the preceding page (Fig. 1.1) but to understand the actual mechanism with more clarity , it become essential to delineate the electrical scenario also on the horizontal plane –.

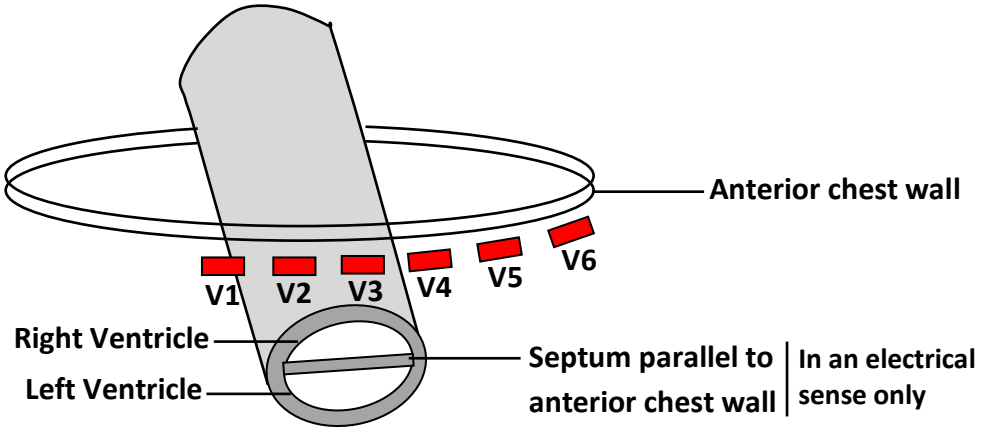
The presence of an **r'** wave in lead V1 is seen specially in the context of this SI SII SIII syndrome due to somewhat delayed activation of the hypertrophied or strained right ventricle. This makes the morphology of V1 pattern as rSr'. In other words this r' denotes the delayed activation of the right ventricle .

There may be a clockwise rotation observed with precordial leads . This entire electrocardiological episode in context with this syndrome can be explained by a single factor , being operated through both the planes – RVH or its acute strain :

Frontal plane reflects the orientation of electrical axis more towards right and upward, attributed to somewhat delayed depolarization of the hypertrophied or strained right ventricle.

Horizontal plane The initial r wave in V1 is followed by an S wave – as a mirror reflection of the left ventricle depolarization and is further followed by a next r' wave due to the delayed right ventricular activation (This r' wave is non-specific finding , not always present).

Display of SI SII SIII syndrome over the horizontal plane as r' in V1 with clockwise rotation –
— In true sense it is electrical translation of mechanical remodelling of RV:



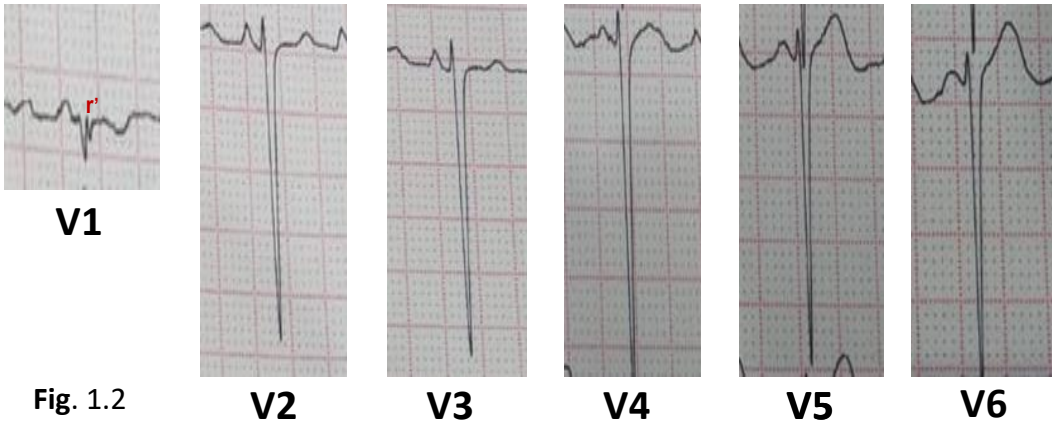


Fig. 1.2

Inscription of r' in V1 with associated clockwise rotation

3. SI SII SII Syndrome is the electrical translation of mechanical remodelling in RV

☐ **Over the frontal plane**

More electricity production by such hypertrophied or strained right ventricle

↓

Pushing the electrical axis rightward and upward

↓

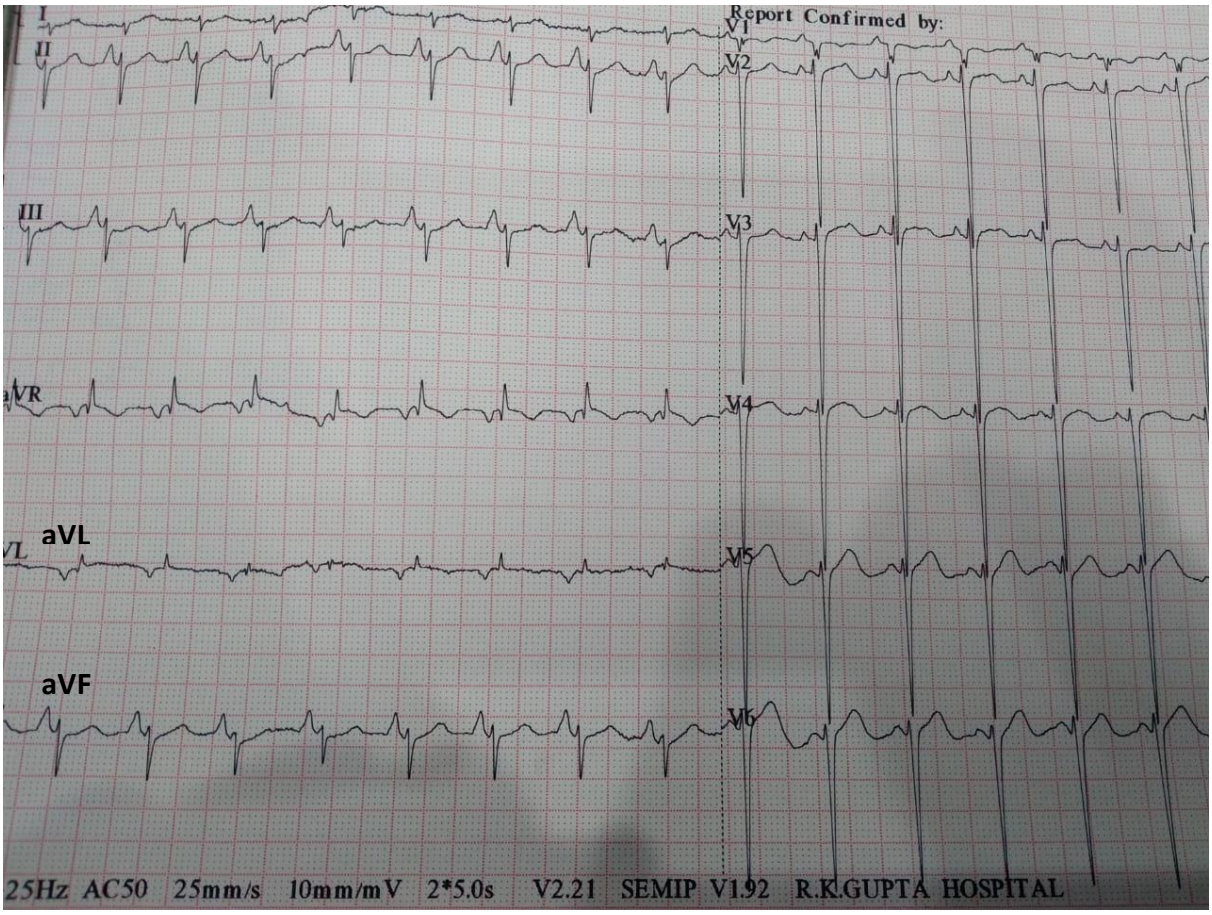
Prominent S-waves in leads I II III with a positive deflection in aVR (qR)
(this entire incidence has been illustrated as such in Fig. 1.1)

☐ **Over the horizontal plane**

With clockwise rotation around the oblique axis , the hypertrophied or strained right ventricle is assumed to adopt a more anterior postion so that the interventricular septum lies parallel to anterior chest wall. This would mean that all or most of the precordial leads – leads V1 to V5 or V6 would reflects rS complexes ——
but in true sense it is the electrical translation of mechanical remodelling of hyperophied or strained right ventricle

4. Illustration by ECG with its full explanation

This ECG belongs to 55 years male with the history of breathlessness since last 25 years



Global Heart Rhythm Forum , posted by Dr. R.K Gupta , Senior consultant Physician , Yamunanagar (Haryana) on 31st March 2024

Findings :

☐ **Frontal plane**

(i) SI SII SIII pattern as evident by prominent S-wave in leads I II III with a positive deflection in lead aVR (qR) – rightward and superior shift of the electrical axis

(ii) The P-pulmonale in leads II III and aVF with P-axis = $+105^0$ (Rt shift of P-axis)

☐ **Horizontal plane**

(i) Clockwise rotation as rS extending from V1 to V6

(ii) The presence of r' wave in V1

(iii) The amplitude of P wave > 1.5 mm, most prominent in V1 to V3

Discussion

- P-pulmonale (P-waves > 0.25 mV) in leads II , III , aVF
- P-axis + 105 P > 1.5 mm in V1-V3

= Right Atrial Enlargement

- Clockwise rotation with R/S ratio < 1 in lead V5 and V6
- SI SII SIII pattern , as discussed

= Right Ventricular Hypertrophy

As per Chou's criteria , this proves to be a case of COPD (Chronic obstructive pulmonary disease)

According to Chou , COPD is likely to be present if one or more of the P wave changes with one or more of the QRS changes as enumerated below , are present on ECG:

P wave changes

1. P waves>0.25 mV in lead II,III,aVF.
2. P wave axis to the right of 80 degrees in the frontal plane.
3. Lead I sign with an isoelectric P wave,QRS amplitude <0.15 mV, and T wave amplitude <0.05 mV.

QRS changes

4. QRS amplitude in all limb leads < 0.5 mV
5. QRS axis to the right of 90 degrees in the frontal plane.
6. QRS amplitude <0.5mV in lead V5 or V6; or R wave <0.7mV in lead V5 or R wave <0.5 mV in lead V6.
7. R/S ratio <1 in lead V5 or V6.
8. $S_1S_2S_3$ pattern with R/S ratio<1 in leads I, II and III.

5. Aetiological factors leading to SI SII SIII Syndrome

The SI SII SIII Syndrome might be reflected as de novo under the following circumstances :

- As a normal variant in children due to right ventricular dominance

- As an expression of right ventricular hypertrophy such as with congenital heart disease or Acquired heart disease i.e COPD .
  It is a known fact that the SI SII SIII syndrome is not an uncommon electrocardiographic finding associated with acquired right ventricular enlargement due to chronic obstructive pulmonary disease .

- Apical myocardial infarction
  The infarction at the apex can cause abnormal conduction , leading to the electrical axis shifting more towards the right side of the heart , producing prominent S-wave in limb leads. In some cases the apical myocardial infarction can lead to increased strain on the right ventricle , most probably due to secondary defects , such as increased pressure in the pulmonary circulation (Pulmonary hypertension) with shifting of the electrical axis towards the right

- The straight back syndrome (straightening of upper dorsal spine due to loss of anterior concavity) - a congenital osseous manifestation without any heart abnormality.

6. Take Home Message

- ☐ This SI SII SIII syndrome seems to be related to somewhat delayed right ventricular depolarization due to remarkable right ventricular hypertrophy or secondary to its acute strain, shifting the electrical axis more right and upward .
  Only simple RVH or its strain might have produced only rightward shift , not so directed upward
- ☐ Prominent S waves in leads I II III with a positive deflection in lead aVR ------- hence the nomenclature SI SII SIII Syndrome
- ☐ There might be clockwise rotation on the horizontal plane. The QRS duration is normal
- ☐ The terminal QRS over the horizontal plane might be associated with a small r' deflection in lead V1. This is non-specific finding , not always present.
- ☐ This syndrome is not a specific marker for a single entity as such but rather indicative of right ventricular overload or strain. It is not an uncommon electrocardiographic finding , seen usually associated with chronic pulmonary disease (eg. COPD)
- ☐ In true sense SI SII SIII syndrome is the electrical translation of mechanical remodelling of hypertrophied or strained right ventricle
- ☐ The presence of such findings on ECG prompts further investigations directed towards the underlying cause , those including echocardiography , chest radiology or at times even CT imaging to access the right heart and pulmonary vasculature.

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VERTICAL P-WAVE AXIS
A WORTH KNOWING FACT

ECG

VERTICAL P-WAVE AXIS : A WORTH KNOWING FACT

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OUTLINE

Introduction (fundamentals)

A vertical P-wave axis > $+60^0$ on the frontal plane specially over 45 years of age has been claimed to be highly suggestive of COPD with emphysema by many studies made so far.

Mechanism of Vertical P-wave axis

- Right atrium is firmly anchored to the underlying diaphragm by a dense pericardial ligament around the inferior vena cava.
- A downward pushing of diaphragm by the hyperinflated lungs occurs in a progressive manner.
- Thus , the right atrium is distorted and depressed inferiorly and vertically with its axis on the frontal plane (>$+60^0$)– known as P-wave axis verticalization on ECG.

Determination of Vertical P-wave axis

Vertical P-wave axis (>$+60^0$) can be accessed by either of the following methods :

- **Bipolar lead system : P-wave amplitude in lead III is greater than in lead I.**
- Unipolar lead system : A predominantly negative P-wave in lead aVL.

Illustration by ECG

Discussion

Take Home Message

References

Vertical P-wave axis : A worth knowning fact

A Narrative Review

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Verticalization means authoritative pin-pointing decision to a concrete solution of a problem as if it is the judiciary stamp put over the decisive court order. The concept of electrical forces generated by the heart also provides immediate access to the electrocardiological principle dealing with its data-analysis tailored towards its utility in clinical practice.
Each cardiac wave P-QRS-T is having its separate electrical axis with a separate clinical sense. P-wave axis on hexagonal lead system is mainly assigned to the right atrium and it may move to a value of $>+60^0$ – this is known as Vertical P-wave axis which is used as a screening tool in a COPD patient with overinflated lungs.

- ☐ **Progressive hyperinflated lungs in COPD push the diaphragm downward with its flattening and the right atrium is also pulled down with a significant rightward deviation.**
- ☐ **Vertical P-wave axis serves as a pointer to access COPD with quantification of its severity as well.**

vertical P-axis is proposed as a 'lone' standard diagnostic and prognostic pointer in a COPD patient over 45 years of age.

1. Introduction (fundamentals)

- **Anatomically** , right atrium lies anterior to the left atrium. The right atrium depolarizes first , inscribing the P-wave on the frontal plane reflecting the dominant vector of right atrial depolarization with its axis usually directed inferiorly with the positive deflection in inferior leads II, III and aVF , and 12-lead electrocardiogram (ECG) can be used to analyse the P-wave in the limb leads with reference to hexagonal lead system.
  (**NB** : And over the horizontal plane the right atrium depolarizes towards V1 , tending to cause an initial positive deflection , and the left atrium depolarizes later and posteriorly , tending to cause a terminal negative P deflection).

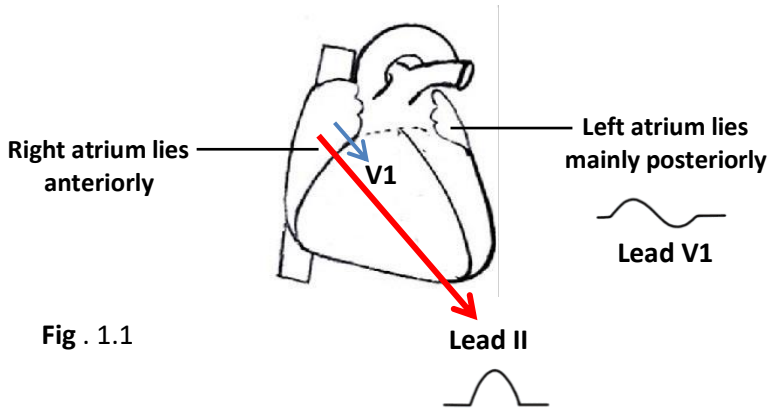


Fig . 1.1

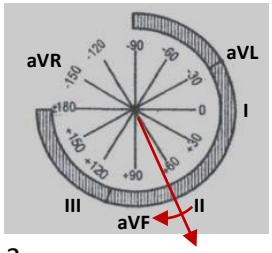
- 

Fig . 1.2 P-wave axis (>+60⁰) Hexagonal lead system (Frontal plane)

In a patient with COPD , the frontal P-wave axis firstly tends to be shifted vertically towards the right as per severity of the illness. Somewhat a vertical P-wave axis > $+60^0$ on the frontal plane specially over 45 years of age has been claimed to be highly suggestive of COPD with emphysema by many studies made so far.

- The main cause of P-wave axis verticalization is overinflated lungs as encountered in COPD patients – such patients have evidence of both emphysema (air space destruction with trapping of air inside) and chronic bronchitis.
- The vertical P-wave axis (> $+60^0$) during a sinus rhythm can be easily accessed at a glance on ECG (the way of determining P-wave axis has been separately discussed on page 32).
- There is a correlation between degree of vertical P-wave axis and the severity of COPD. The quantum of P-wave verticalization crawling rightwards is also correlated inversely with FEV1 (pulmonary function test). And there have been its significant correlation also with the radiological severity of the emphysematous changes in COPD.
- **The determination of P-wave axis on ECG may prove itself as a quick provider for the earlier diagnosis of COPD with emphysema.**

2. Mechanism of Vertical P-wave axis

Right atrium is firmly anchored to the underlying diaphragm by a dense pericardial ligament around the inferior vena cava. A downward pushing of the diaphragm by the hyperinflated lungs occurs in a progressive manner. Thus , the right atrium is distorted and depressed inferiorly and vertically with its axis on the frontal plane (>$+60^0$)– known as vertical P-wave axis on ECG.

The entire concept is illustrated by the following sketch :

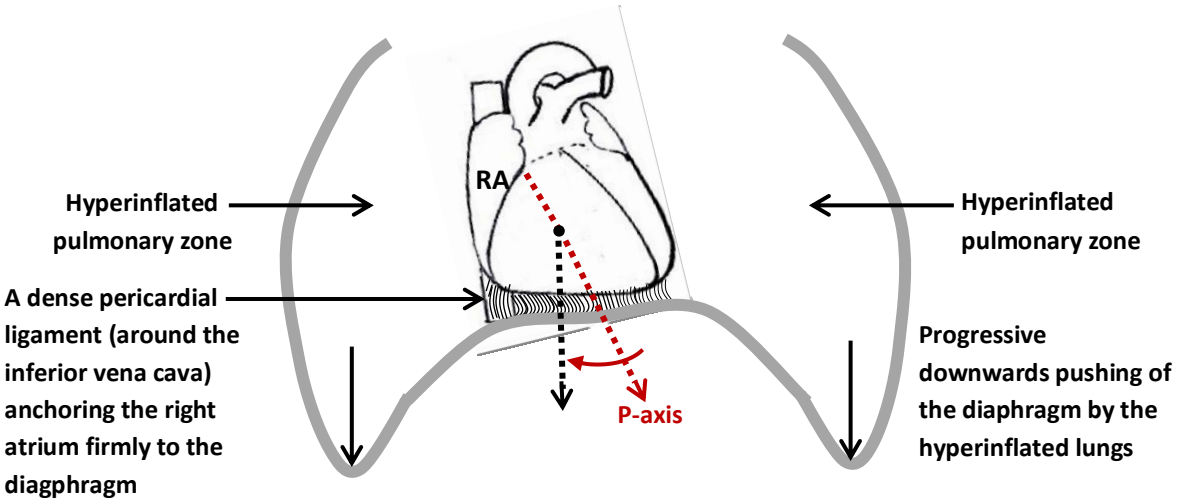


Fig . 1.3

3. Determination of Vertical P-wave axis

The outstanding electrophysiological principle

> Any exploring lead placed within a range of 90^0 in respect to cardiac vector records positive current , at 90^0 equiphasic deflection or no deflection and beyond 90^0 negative deflection.

This law is applicable to any wave – P-QRS-T on hexagonal lead system with reference to 12-lead ECG. Therefore , it is also applicable to P-wave while determining its axis verticalization. And accordingly the hexagonal lead system can be dissected into two zonal systems :

- **Bipolar lead system with respect to lead I and lead III**.
- **Unipolar lead system with respect to lead aVL and aVF.**

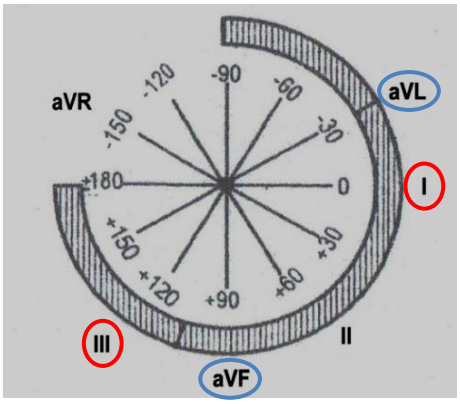


Fig . 1.4 **HEXAGONAL LEAD SYSTEM**

Vertical P-wave axis (>+60^0) can be accessed by either of the following methods :

- **Bipolar lead system** : P-wave amplitude in lead III is greater than in lead I.
- **Unipolar lead system** : A predominantly negative P-wave in lead aVL.

Out of these two criteria , the bipolar lead system is considered to be more sensitive. A recent study done by Puneet Gupta et al. in year 2021 shows that the bipolar lead system is a more sensitive marker of determining P-wave axis in emphysema compared to the unipolar lead system (sensitivity 88% vs 66% respectively). This study excluded patients younger than 45 years because the vertical P axis may be a normal finding in this age group.

Reference :
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https://wjgnet.com/1949-8462/full/v13/i10/533.htm

The changes in the frontal plane P-wave axis as discussed above are one of the most important exciting changes noted on the standard 12-lead ECG system with emphysema.

4.Illustration by ECG

Vertical P-wave axis (P axis >+60^0) serves as a useful index towards diagnosing emphysema , as associated with COPD.

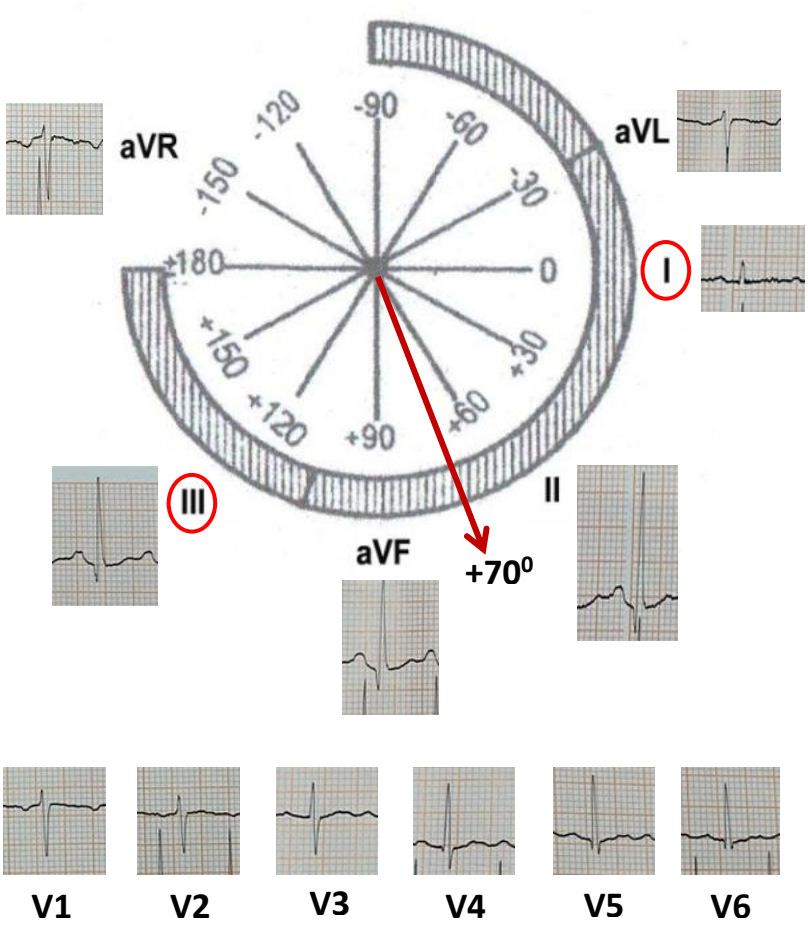

Fig . 1.5

Source : CME India dated 20 Feb 2023 by Dr Neeraj DNB Med , Sonipat, HARYANA (He also sent the details of this case personally to me as well)
Arranging the ECG tracings on hexagonal lead system for the purpose of better understanding

History : A known case of COPD

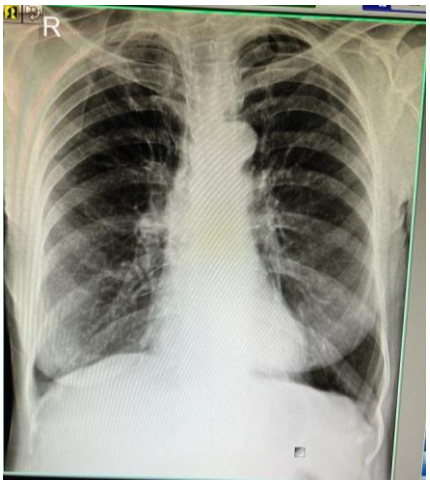

Fig . 1.6

Discussion :

- **Bipolar lead system** : P-wave amplitude is greater in lead III than in lead I
- Unipolar limb system : P wave negative in aVL
- The calculated P axis = approx +70^0
  (P-wave axis verticalization >+60^0 is +nt here)
- Chest radiology -hyperinflated lungs , vertical tubular heart and the flattened diaphragm are well correlated with the P-wave axis verticalization in this case.

NB : The main QRS axis is also projected on the hexagonal lead system (approx +70^0) having its largest deflection in inferior leads **(the maximum deflection is noted in leads , nearest to the vector).**

5. Discussion

P-axis verticalization serves as a very useful tool toward diagnosing COPD with emphysema. This phenomenon has been described by so many workers - to mention a few - Spodick's report in 1959 with a vertical P-wave axis > $+80^0$ in COPD was the important observation. Again in 1999, Balijepally and Spodick also noted P-wave axis >$+70^0$ with a sensitivity of 89% and a specificity of 96% in COPD. **It would be worthwhile to mention here that P-wave changes in COPD are the result of right atrial downward displacement by hyperinflated lungs ± right atrial enlargement**. And possibly the workers under discussion had included both these two factors responsible for Vertical P-wave axis.

As per newer observation Vertical P-wave axis is considered positive even when it is more than $+60^{0.}$. The changes in the frontal P-wave axis are among one of the most important changes noted on the 12-lead ECG system in COPD with emphysema. In most of normal individual , the frontal P-wave axis is found to be lying within a very narrow range $+45^0$-60^0. Further to say , in patients with COPD the frontal P-wave axis is shifted vertically / rightward in a range of $+60^0$ or more. **A vertical P-wave axis (>$+60^0$) on the hexagonal lead system specially in individuals above 45 years of age has been considered highly suggestive of COPD with emphysema** by several studies done so far.

The prevalence of emphysema in such COPD patients with vertical P-wave axis >$+60^0$ has been found to be higher than in the control group : 85% vs 4.4% with a remarkable sensitivity and specificity (94.76% and 96.47% respectively). The severity of illness is also dependent upon the degree of P-wave axis verticalization – and if it is >$+75^0$, it points toward a very severe degree of such illness.

6. Take Home Message

- ☐ Right atrium is firmly anchored to the underlying diaphragm by a dense pericardial ligament around the inferior vena cava. A downward pushing of the diaphragm by the hyperinflated lungs occurs in a progressive manner. Thus , the right atrium is distorted and depressed inferiorly and vertically with its axis on the frontal plane (>$+60^0$)– known as vertical P-wave axis on ECG.
- ☐ Vertical P-wave axis (>$+60^0$) can be accessed by either of the following methods :
  - Bipolar lead system : P-wave amplitude in lead III is greater than in lead I.
  - Unipolar lead system : A predominantly negative P-wave in lead aVL.
- ☐ The determination of P-wave axis on ECG may prove itself as a quick provider for the earlier diagnosis of COPD with emphysema.
- ☐ There is a correlation between degree of vertical P-wave axis and the severity of COPD. The quantum of P-wave verticalization crawling rightwards is also correlated inversely with FEV1 (pulmonary function test). And there have been its significant correlation also with the radiological severity of the emphysematous changes in COPD.

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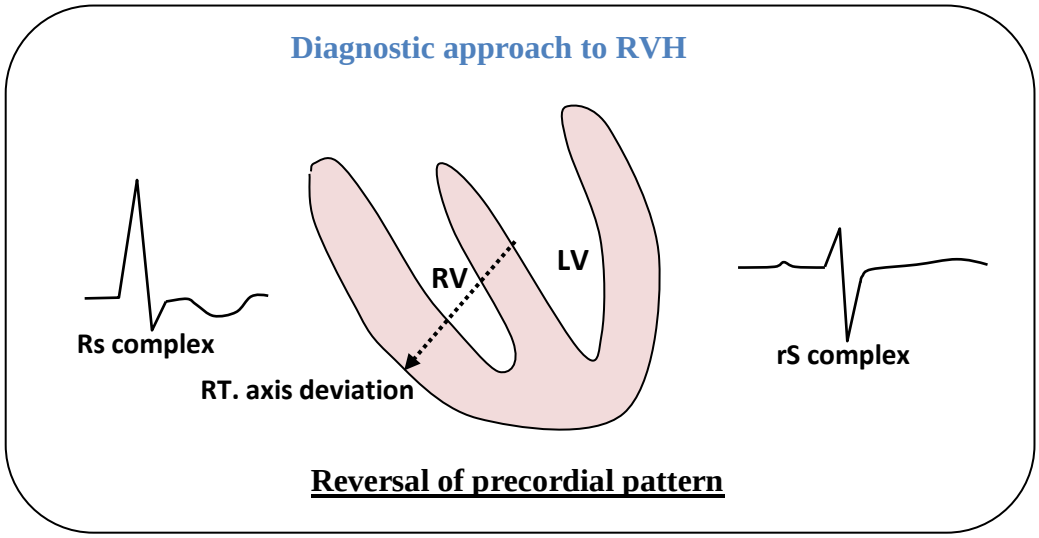
A USEFUL ADDENDUM

- RVH
- RAE
- RBBB

ECG changes in right ventricular hypertrophy



ECG features for RVH on ECG

☐ **Diagnostic criteria (Reversal of precordial pattern)**

- Dominant R wave in V1 (> 7mm tall or R/S ratio > 1) with peak time is typically prolonged (35-55 ms)
- Dominant S wave in V5 or V6 (> 7mm deep or R/S ratio < 1).
- Right axis deviation of +110° or more in addition (Virtually the electrical axis is always shifted to the right – almost mandatory)

☐ **Supporting criteria**

- Right atrial enlargement - P-pulmonale : right atrial dilatation / overload.
- Secondary changes : ST depression / T-wave inversion in the right precordial (V1-V2 ,at times V3) and inferior (II , III , aVF) leads due to repolarization abnormalities of the right ventricular myocardium / subendocardial ischmeia.
- Deep S wave in the lateral leads (I , aVL , V5-V6) – mirror effect of RVH
- Dominant S wave is lead I , II and III (S1 S2 S3 syndrome associated with axis deviation superiorly and to the right towards the 'north-west' region)

☐ **Other abnormalities such as**

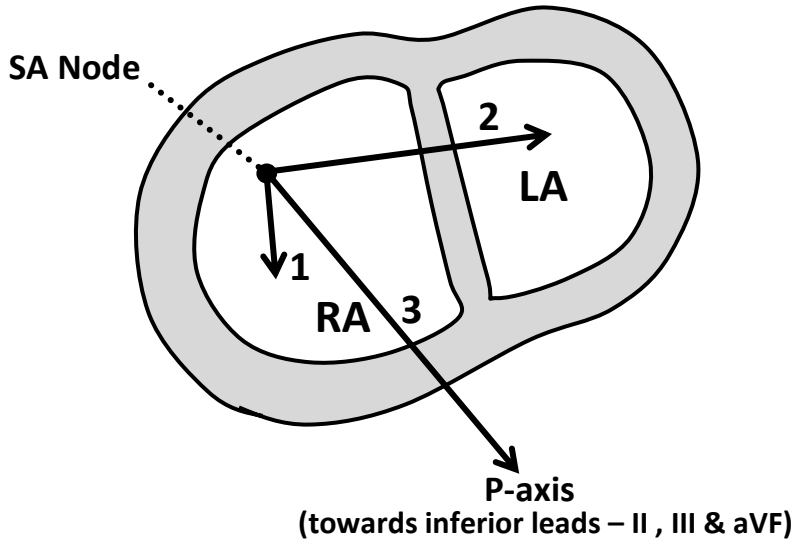
- Right bundle branch block (complete or incomplete). It signifies dilatation or overload of the right ventricle.

NB : There is a lack of accepted criteria to diagnose RVH in the presence of RBBB. The presence of tall R wave in V1 in association with right axis deviation of 110^0 or more with supporting criteria would be considered as suggestive of RVH.

ECG Criteria of Right Atrial Enlargement

With right atrial enlargement , the amplitude of the initial component of P-wave increases , i.e. the overall amplitude of P-wave in lead II and the amplitude of inital component of P in lead V1. The width remains unchanged because the terminal portion of the P-wave is left atrial in origin.

Right atrial enlargement also makes the right atrium to dominate over the left atrium from the electrical point of view , the P-axis is deviated clockwise towards more than $+70^0$.



The ECG changes suggestive of right atrial enlargement are mentioned as below :

- > 2.5 mm in the inferior leads (II, III and AVF) , known as **P-pulmonale**.
- > 1.5 mm in V1 and ±V2

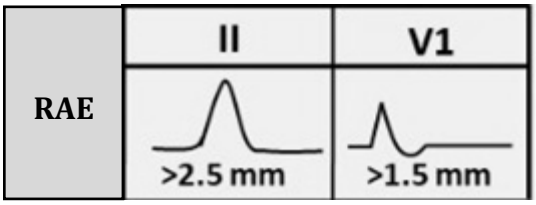


Fig . 1.5

- P-wave axis in the frontal plane is more than $+70^0$.

The ECG changes suggestive of right atrial enlargement correlate poorly with its clinical and anatomic findings

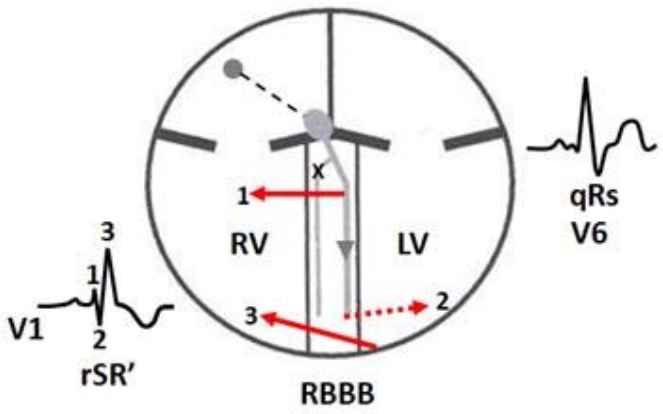
- Better criteria can be laid down from the QRS complex since QRS changes may occur due to high incidence of RVH in association with RAE.
    - QR, Qr, qR, or qRs morphology in lead V1 (in absence of coronary heart disease)

      This is to be noted that Q/q is the septal wave which is directed towards the left ventricle , away from the right ventricle.

Ref : ECG Learning Centre (Created by Eccles Health Science Library)
https://ecg.utah.edu/lesson/7

ECG Changes in Right bundle branch block

☐ With the blockade of Right bundle branch (RBBB) :
Septal depolarization from left to right , and then the subsequent activation of the right ventricle via the left ventricle



☐ Criteria for RBBB

Leads oriented to the right ventricle , e.g. lead V1	**Leads oriented to the left ventricle , leads V5 ,V6 and lead V1**
1. An initial small r wave , due to left septal depolarization	1. A small initial q wave , due to left septal depolarization
2. This is followed by an S, or more likely an s, wave which is due mainly depolarization of the left free wall. This S wave is attenuated and may even disappear completely.	2. There is a relatively tall R wave, due mainly to repolarization of the left free wall.
3. There is a terminal bizarre and slurred R wave (R') due to late and anomalous right septal and right free wall depolarization.	3. There is a terminal bizarre and slurred S wave due to late and anomalous right septal and right free wall activation.

NB : The QRS duration is increased to 120 ms or longer in complete RBBB.

(INCOMPLETE RBBB : The conduction through right bundle branch and its ramification is delayed in incomplete RBBB.

- The WHO/ISFC task force criteria for incomplete RBBB are the same as for complete RBBB, except the QRS duration is < 120 ms)